A Time Trend Study of Significantly Elevated Perfluorocarboxylate Levels in Humans after Using Fluorinated Ski Wax

HELENA NILSSON,*,† ANNA KÄRRMAN,† HÅKAN WESTBERG,†,‡ ANNA ROTANDER,† BERT VAN BAVEL,† AND GUNILLA LINDSTRÖM†

Man–Technology–Environment (MTM) Research Centre, Örebro University, SE-701 82 Örebro, Sweden, and Department of Occupational and Environmental Medicine, Örebro University Hospital, SE-701 85 Örebro, Sweden

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A time trend study focusing on ski waxing technicians' exposure to perfluorinated chemicals (PFCs) from fluorinated wax fumes was performed in 2007/2008. Levels of eight perfluorocarboxylates and three perfluorosulfonates were analyzed in monthly blood samples from eight technicians. Samples were collected before the ski season, i.e., preseason, then at four FIS World Cup competitions in cross country skiing, and finally during an unexposed 5-month postseason period. The perfluorinated carboxylates perfluoroheptanoic acid (PFHpA), perfluorooctanoic acid (PFOA), perfluorononanoic acid (PFNA), perfluorodecanoic acid (PFDA), and perfluoroundecanoic acid (PFUnDA) bioaccumulate, and continued exposure may contribute to elevated levels in ski technicians compared to the general population. The wax technicians' median blood level of PFOA is 112 ng/mL compared to 2.5 ng/mL in the general Swedish population. A significant correlation was found between number of working years and levels of perfluorocarboxylates. The PFOA levels in three technicians with "low" initial levels of PFOA (<10.0 ng/mL in preseason blood) increased by 254, 134, and 120%, whereas five technicians with "high" initial levels (>100 ng/mL in preseason sample) were at steady state. PFHxA is suggested to have a short half-life in humans relative the other perfluorocarboxylates. The levels of perfluorosulfonates were unaffected by the wax exposure.

Introduction

Fluorinated ski waxes are applied to the skis' soles by using heat of approximately 130–220 °C (*1*–*4*). During this process airborne particles and fumes containing a blend of gaseous organofluorine compounds are emitted (*5*, *6*). Inhalation of thermal degradation products from fluoropolymers could cause alveolic edema (*7*); polymer fume fever, informally called the Teflon flu (*8*, *9*); severe dyspnea; decreased pulmonary function (*10*); and respiratory distress syndrome (*11*).

In general, two types of ski wax are used: grip and glide wax. Grip wax is used to let the skis' sole make contact with the snow and allow the skier to kick in a forward motion. This type of wax does not contain fluorinated additives. On the other hand, fluorinated organic components are added to most glide waxes due to their unique surfactant properties. The waxes create oil- and water resistance to surfaces as well as preventing adhesion of snow, ice, and dirt that slow down the skis' movement. The exact composition of fluorinated additives are rarely disclosed by producers. However, most gliding waxes are petroleum-derived products $CH_3(CH_2)_nCH_3$, where n varies from 10 to 80, mixed with semifluoroalkanes $CH_3(CH_2)_m(CF_2)_{m'}CF_3$, where m varies from 14 to 20 and m' from 2 to 16 (*12*, *13*). Waxes are available in many different formulas to match the temperature and snow conditions, which have resulted in numerous US patents (*14*, *15*). The estimated annual production of ski wax is 275 tonnes globally, based on information from a major producer (*16*).

No data on perfluorinated chemical (PFC) blood levels in ski wax technicians exist in the international literature. However, perfluorinated chemicals have been used for many years (*17*) in a number of products, and consequently, there is an environmental background exposure to general populations as well as individual exposure due to applications of fluorinated chemicals (*18*–*20*).

Several PFCs have been found in blood (*21*, *22*), serum (*23*), liver (*24*), breast milk (*25*), and umbilical cord blood (*26*) from general populations worldwide. Levels of perfluorooctanoic acid (PFOA) in the general Swedish population range between 0.5 and 12.4 ng/mL whole blood, with an average of 2.5 ng/mL (*27*). In the US population, Calafat et al. reports the range of PFOA from 2.4 to 2.9 ng/mL (*28*). Developmental toxicity (*26*, *29*) and hormonal disruption (*30*) have been associated with PFOA in humans, and reduced fecundity was recently reported at PFOA and perfluorooctane sulfonate (PFOS) plasma levels present in the general population (*31*).

The study focused on cross country skiing wax technicians' exposure to PFCs and the temporal changes of perfluorochemicals in their blood. We measured 11 PFCs in monthly blood samples from eight technicians collected during 10 months, i.e., preseason, during the course of 2007/2008 FIS World Cup competitions, and the subsequent unexposed 5-month period.

The aim of the study was to determine if persons frequently exposed to fluorinated ski waxes have elevated blood levels of PFCs compared to the general population. Furthermore, we aimed to assess the temporal trends of the levels over one year covering the exposed period of cross-country World Cup season 2007/2008 as well as the unexposed months before and after the ski season.

Experimental Section

Study Participants. The ski wax technicians (n = 8) are employed by the Swedish and US national cross-country ski teams. Seven technicians are Swedish and one is Estonian. They are 27–51 years old, and worked as wax technicians 3–15 years. The technicians' years in the profession, team affiliations, nationalities, and ages are presented in Table 1. Seven technicians are seasonally employed during the World Cup ski season and have other occupations from March to December. Technician 3 is a full-time employee at the Swedish Ski Federation. During the exposed skiing season from December to March the technicians apply fluorinated ski wax for approximately 30 h a week. None of the participants are blood donors or smokers.

Written informed consent was obtained from all participants at recruitment. The ethical vetting board of Uppsala,

* Corresponding author phone: +46 19 30 12 09; fax: +46 19 30 35 66; e-mail: helena.nilsson@oru.se.
† Örebro University.
‡ Örebro University Hospital.

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TABLE 1. Years as Wax Technician, Team Affiliations, Nationalities, Ages, and Occupations of the Ski Wax Technicians (n = 8)

	December to March (exposed period)				
technician	years as wax technician	team	nationality	age	April to November (unexposed period) occupation
1	3	Sweden	Swedish	27	student
2	6	Sweden	Swedish	51	carpenter
3	6	Sweden	Swedish	33	full-time employee at Swedish Ski Association
4	10	Sweden	Swedish	38	gardener
5	5	Sweden	Swedish	32	fire fighter
6	15	US	Swedish	45	carpenter
7	7	US	Estonian	30	carpenter
8	12	US	Swedish	45	engineer

TABLE 2. Ranges of Perfluorinated Carboxylates (C4–C11) in Whole Blood (ng/mL) of Wax Technicians (n = 8)[a]

technician		PFBA	PFPeA	PFBuS	PFHxA	PFHpA	PFHxS	PFOA	PFNA	PFOS	PFDA	PFUnDA
1	A	0.12	<0.06	<0.02	<0.07	0.82	<0.30	4.8	0.86	0.28	0.87	0.11
	B	0.33–0.68	<0.06–0.14	<0.02–0.03	2.4–12	1.2–6.6	<0.30	6.3–17	0.76–2.2	0.30–0.39	1.1–2.3	0.14–0.39
	C	0.16–0.57	<0.06	<0.02	<0.07	5.7–7.3	<0.30	17–20	1.7–2.8	0.34–0.36	1.8–3.7	0.30–0.46
2	A	0.09	<0.06	<0.02	<0.07	<0.37	1.9	8.50	3.8	24	1.9	0.18
	B	0.18–0.23	<0.06–0.07	<0.02	<0.07–4.8	<0.37–2.5	1.6–2.1	10–20	3.6–5.7	23–26	2.4–3.3	0.36–0.55
	C	<0.08–0.10	<0.06	<0.02–0.04	<0.07–0.09	<0.37–1.7	3.3–4.3	19–23	6.1–7.1	21–25	3.5–4.8	0.31–0.47
3	A	0.26	<0.06	<0.02	<0.07	1.0	1.4	151	14	14.9	8.84	0.79
	B	0.11–0.23	<0.06–0.10	<0.02	0.33–5.4	1.5–4.8	0.78–1.5	146–150	15–18	12–14	9.2–20	1.0–2.2
	C	<0.08	<0.06	<0.02	<0.07–1.2	0.87–2.1	0.96–3.3	134–153	15–20	12–17	11–24	1.1–2.5
4	A	0.11	<0.06	0.030	<0.07	0.76	1.70	127	21	13	6.8	1.0
	B	0.09–0.40	<0.06	<0.02	0.22–2.2	1.3–3.7	1.7–1.9	114–131	18–21	11–13	6.6–8.7	0.73–1.2
	C	<0.08	<0.06	<0.02	<0.07	1.0–3.4	1.6–2.0	101–122	17–19	10–11	8.2–9.8	1.2–1.4
5	A	0.09	0.080	<0.02	<0.07	<0.37	1.6	10	1.7	14	1.0	0.35
	B	0.20–0.46	<0.06–0.07	<0.02	0.68–5.2	0.75–6.3	1.5–1.8	12–22	2.0–3.7	14–15	1.6–3.7	0.38–0.75
	C	–	–	–	–	–	–	–	–	–	–	–
6	A	…	…	…	…	…	…	…	…	…	…	…
	B	0.38–1.1	0.12–0.14	<0.02	0.08–0.80	12–12	2.0–2.4	253–276	56–65	24–26	20–22	2.4–2.8
	C	<0.08	<0.06	<0.02	<0.07–0.08	6.9–10	1.2–1.3	249–268	45–57	22–27	9.1–10	1.2
7	A	–	–	–	–	–	–	–	–	–	–	–
	B	0.12–0.32	<0.06–0.07	<0.02	0.14–0.65	0.68–2.4	0.69–0.82	100–106	10–12	8.1–8.2	6.1–7.7	0.60–0.73
	C	–	–	–	–	–	–	–	–	–	–	–
8	A	0.13	0.09	<0.02	0.13	19	1.4	474	145	7.2	11	2.2
	B	<0.08	<0.06	<0.02	0.14–0.71	15–20	1.2–1.6	528–535	133–163	7.0–8.0	10–13	1.4–2.6
	C	<0.08	<0.06	<0.02	<0.07–0.30	7.7–12	1.2–1.9	468–520	104–128	7.0–9.0	7.3–12	0.58–1.8

[a] A = preseason (Sep 2007), B = during World Cup season 2007/2008 (Dec 2007 to Mar 2008), and C = postseason (Apr to Aug 2008). A dash indicates that no sample was provided.

Sweden (Reference No. Dnr 2007/199) and the Swedish Environmental Protection Agency (Reference No. 721-1519-07Mm) approved the study protocol.

Sampling Design. A total of 57 individual blood samples (approximately 8 mL) were collected before (in September 2007), during (at the FIS World Cup competitions 2007/2008), and after (from April to August 2008) the exposed period. Samples were drawn after full working days at World Cup events in Kuusamo, Finland (December 2007) followed by Val di Fiemme, Italy (January 2008), Otepää, Estonia (February 2008), and finally in Holmenkollen, Norway (March 2008). During the unexposed period from April to August 2008 samples were taken in the participants' local hospitals. Blood was drawn from a single point vein puncture and samples were stored in blood tubes containing EDTA at −20 °C prior to analysis.

Chemicals. Details are available in the Supporting Information.

Extraction and Analysis. Levels of perfluorinated carboxylic acids perfluorobutanoic acid (PFBA), perfluoropentanoic acid (PFPeA), perfluorohexanoic acid (PFHxA), perfluoroheptanoic acid (PFHpA), PFOA, perfluorononanoic acid (PFNA), perfluorodecanoic acid (PFDA), perfluoroundecanoic acid (PFUnDA), and perfluorinated sulfonates perfluorobutane sulfonate (PFBS), perfluorohexane sulfonate (PFHxS), and PFOS were analyzed. Detailed information of the sample extraction is described elsewhere (*32*). Briefly, internal standards ($^{13}C_4$-PFOA and $^{13}C_4$-PFOS) and 2 mL of formic acid/water (1:1v/v) were added to 0.5 mL of whole blood. After sonication and centrifugation, the supernatant was extracted using solid-phase extraction with weak anion exchange (Waters Oasis WAX), and the perfluorinated compounds were eluted with 1 mL of 2% ammonium hydroxide in methanol. The volume of the blood extract was adjusted to 200 μL using nitrogen. Performance standard $^{13}C_5$-PFNA and 300 μL of 2 mM ammonium acetate in water were added prior to analysis. Analysis was performed using an Acquity UPLC coupled to a Quattro Premier XE (Waters Corp.) with an atmospheric electrospray interface operating in negative ion mode (ES-MS/MS). Separation was performed on an Acquity BEH C18 2.1 × 50 mm, 1.7 μm kept at 50 °C. A PFCs isolator (Waters Corp.) was inserted between the pump and injector to remove any fluorochemicals originating from the UPLC system. Injection volume was 10 μL and the flow rate was set to 400 μL/min. Details of UPLC gradient program are shown in the Supporting Information. We used multiple reaction monitoring of molecular anion $[M - H]^-$ for carboxylates and $[M^-]$ for sulfonates and measuring the product ions $[M - COOH]^-$ and $[FSO_3]^-$ for carboxylates and sulfonates, respectively.

Quality Assurance. Quantification was performed using the internal standard method using $^{13}C_4$-PFOS for the perfluorosulfonates and $^{13}C_4$-PFOA for the perfluorocarboxylates. All samples were quantified using a seven point calibration curve with a relative standard deviation (RSD) of the relative response factors <15% for all compounds. The

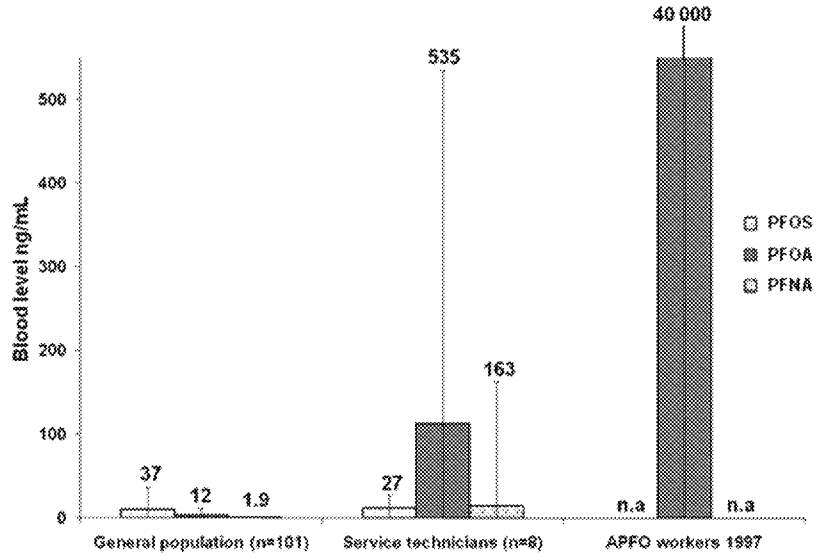

FIGURE 1. Comparison of median whole blood concentrations of PFOS, PFOA, and PFNA in human blood from a general population (n = 66) (*27*), the present study, and 3M workers (*35*). Whiskers show minimum to maximum levels; n.a. = not analyzed.

mean recoveries for internal standards in all samples were 80% for PFOA and 70% for PFOS. Linear and branched PFOS isomers were combined in the quantification. If any of the target compounds were present in the laboratory blank samples, the mean blank signal plus three standard deviations of multiple blank injections were subtracted from the calculated concentrations in the samples. A blank-corrected concentration was only reported if the blank level was equal to or less than 50% of the uncorrected concentration. Recovery, reproducibility, and detection limits (mean blank level + 3 standard deviations) for 11 PFCs in whole blood are given in Table S1 in the Supporting Information. On a regular basis our laboratory takes part in QA/QC studies with good results (z-scores < 2) (*33*).

Statistical Analysis. Spearmans rank correlation test (two-tailed) was performed to assess the relation between PFCs, biological age, and the number of years in the profession.

Results and Discussion

In the present study, PFOA (4.8−535 ng/mL), PFNA (0.8−163 ng/mL), PFDA (0.9−24 ng/mL), PFUnDA (0.1−2.8 ng/mL), and PFOS (0.3−27 ng/mL) were detected in all samples. PFHxS (0.3−4.3 ng/mL) was found in 93% of the samples, i.e., in all except the first four monthly samples from technician 1.

Generally, PFHxA (<0.07−12 ng/mL) was only observed in samples collected during the exposed period from December 2007 to March 2008 and was not found over the detection limit in samples collected during the unexposed months. Concentrations of PFHpA varied between <0.37 and 20 ng/mL and was <LOD (limit of detection) in five samples from technician 2 and technician 5. PFBA, PFPeA, and PFBS were found in 35, 10, and 7 samples, respectively. Ranges of PFC levels from the World Cup season (frequent exposure to fluorinated waxes) and before and after the ski season (unexposed period) are shown in Table 2. The full details of individual levels from all measurements are given in Table S2 of the Supporting Information. For comparison reasons, when referring to PFC levels in studies reporting on a serum basis, these levels have been adjusted by a factor of 2 in order to give the whole blood level, hereby assuming equal distributions (*34*).

Spearmans rank correlation coefficients (r) are presented in Tables 3 and S4 in the Supporting Information. Complete correlation results are available in Table S3 in the Supporting Information.

TABLE 3. Spearmans Rank Correlation Coefficient, r, between Concentrations of PFCs and Biological Age and Number of Years as Professional Wax Technician

	age	years as technician
PFBA	−0.3574[b]	−0.2885[a]
PFPeA	−0.1675	−0.1628
PFBuS	0.0173	−0.0595
PFHxA	−0.1641	−0.2192
PFHpA	−0.0319	0.4071[b]
PFHxS	0.6923[c]	0.2939[a]
PFOA	0.2601	0.7790[c]
PFNA	0.4379[c]	0.9215[c]
PFOS	0.5945[c]	0.1286
PFDS	−0.1921	−0.2786[a]
PFDA	0.2065	0.6713[c]
PFUnDA	0.154	0.6983[c]

[a] P = 0.01−0.05. [b] P = 0.001−0.01. [c] P < 0.001.

To determine if the wax technicians blood levels are significantly different from an unexposed group of men of ages 19−47 years (n = 40), we simply compared the median levels of the two groups (*27*). The median for PFOA in the unexposed group is 2.7 ng/mL compared to 112 ng/mL (mean = 140 ng/mL) for the wax technicians' and 0.2 ng/mL for PFNA for the unexposed group compared to 14.7 ng/mL (mean = 29 ng/mL) for the wax technicians. Three technicians showed initial levels of PFOA <10 ng/mL, but five technicians had levels >99 ng/mL in the preseasonal sample from September 2007. The highest and median PFOA levels in our study (535 and 112 ng/mL) are 43 and 45 times higher than the maximum and median levels found in a study by Kärrman et al (*27*). Five of the eight technicians had PFOA levels almost as high as occupationally exposed workers at a 3M plant (*35*). Figure 1 shows the median levels and range of PFOA, PFNA, and PFOS in the technicians compared to the general Swedish population and occupationally exposed 3M workers.

The carboxylate found at the second highest level was PFNA (10.1−163 ng/mL). We observed PFNA levels 15−270 times higher than in representative populations from Sweden, China, and North America (*36*−*38*). PFHpA was found in 90% of the samples ranging from <0.4 to 19 ng/mL with a median of 2.8 ng/mL. Ericson et al. (*40*) did not find PFHpA in any blood samples (n = 48) from Spain and Calafat et al. (*38*) detected the analyte in 6% of samples (n = 2 094) from a representative US population (mean 0.2 ng/mL). Perflu-

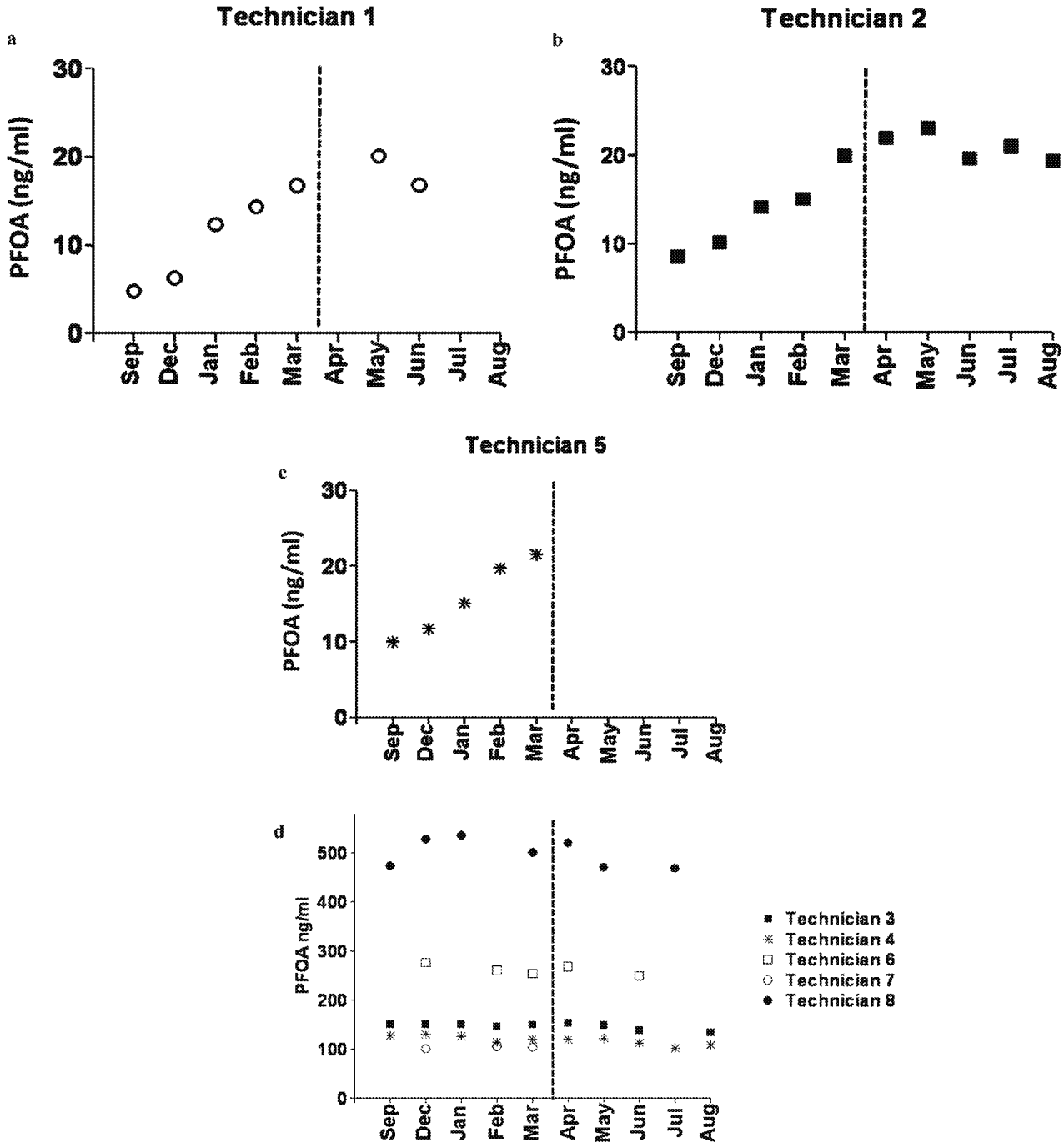

FIGURE 2. Temporal trend of whole blood levels (ng/mL) of PFOA in (a) technician 1, (b) technician 2, (c) technician 5, and (d) technicians 3, 4, 6, 7, and 8. The dotted line marks the end of the World Cup season. Unexposed period (Sep 2007), exposed period (Dec 2007 to Mar 2008), and unexposed period (Apr to Aug 2008).

orinated carboxylates with chain lengths >10 are rarely found in human blood from a general population (*37*–*39*) but we find PFUnDA in 100% of the samples in the range of 0.1–2.8 ng/mL. PFHxA is also seldom detected in human blood, but in our study it was found in all samples collected during the World Cup season but in very few of the samples taken pre- and postseason. The mean level of PFDA is 7.9 ng/mL, which is an elevation of up to 80 times compared to representative populations (*28, 40*). The variation in PFDA levels between the two teams may be explained by different waxes of choice since all teams work independently in testing out the optimum combination of grip- and glide waxes. However, the median levels of PFOS (12.2 ng/mL) and PFHxS (1.64 ng/mL) were comparable to those of general populations worldwide (*21, 27, 40*–*42*).

Generally, levels of carboxylates PFOA, PFNA, PFDA, and PFUnDA decreased with increasing carbon chain length in samples collected during the exposed period.

Time Trends. The PFOA levels in technicians 1, 2, and 5, who have low initial levels of PFOA (<10.0 ng/mL in sample from Sep 2007) increased by 254, 134, and 120%, whereas the five technicians with higher initial levels (>100 ng/mL) increased by 6–29% from September 2007 to March 2008, i.e., during the course of skiing season. The temporal trend for PFOA concentrations is presented in Figure 2, which shows that the highest PFOA levels for technicians 1 and 2 are in samples from April or May, although the exposure ended in March.

This delay in dose–response might suggest a toxicokinetic lag time or metabolization of PFOA precursors following the

exposure of fluoroorganic compounds released during waxing (*5*, *6*). The precursor molecules have not been identified in this study nor is it known if they are present in the ski wax as a byproduct or are formed during the process of heating the wax upon application. Previous studies have shown that precursors like FTOHs, fluorotelomer unsaturated acids (FTUCAs), and polyfluoroalkyl phosphate surfactants (PAPs) can biologically degrade to PFOA and PFNA (*43*–*47*). It is likely that this metabolization also occurs in humans, but to date, there are no publications on this available in the international literature. It is of great importance to both the public and legislators that the routes and magnitude of direct and indirect exposure to PFCAs are properly identified.

From April or May and throughout the unexposed period there was stabilization in PFOA concentrations for all technicians, regardless of initial concentration. It seems like the PFOA levels have reached a steady state for technicians 3, 4, 6, 7, and 8, who had initial levels exceeding 100 ng/mL, since their perfluorocarboxylate concentrations do not increase over time despite experiencing exposure to the same waxes and fumes as the technicians who increase by 10-fold. It still remains to be elucidated whether a reservoir in the human body accumulates excess amounts of perfluorocarboxylates after a threshold level in the blood is reached and how specific interactions or exchange between the organs occur. Elevated levels of PFHxA, compared to the whole sampling campaign, were seen in the December 2007 samples, especially for technicians 1–5 (Tables 2 and S2 in the Supporting Information) ranging between 0.65 and 0.80 ng/mL and 2.23 and 12.20 ng/mL from US and Swedish teams' technicians, respectively. Those samples have been reanalyzed and any analytical or instrumental errors can be ruled out. Gannon and colleagues (*48*) report PFHxA to be eliminated rapidly, within 24 h, and almost entirely in its unmetabolized form when excreted in the urine of rats. Our findings suggest a fast elimination also in humans. Since the sampling interval is 4 weeks and the PFHxA levels decrease <LOD for most technicians in that period of time, it is not possible to calculate an exact terminal half-life. We propose a half-life <4 weeks for PFHxA. To our knowledge, this is the first time a suggested short half-life of PFHxA in humans is reported in the international literature. Levels of the perfluorosulfonates remain stable throughout the sampling campaign for all participants.

Correlations. The relation between a technicians' PFOA level and the number of years in the profession is visualized in Figure 2 with the three least experienced technicians (≤6 years) showing the lowest levels of PFOA. However, technician 3, who has also been in the profession for 6 years, showed a median level of 150 ng/mL, which is not consistent with the above-mentioned findings. This participant may have been more frequently exposed to ski wax, since he has been full-time employed by the Swedish Ski Federation for the last 2 years, unlike the other technicians, who have only worked during the ski season. Spearman's rank correlation test (coefficient, r) was used to relate the blood levels of PFCs to biological age and the number of working years in the ski industry. A significant correlation ($P < 0.01$) was found between the number of years in the profession and levels of PFBA ($r = -0.29$), PFHpA ($r = 0.41$), PFOA ($r = 0.78$), PFNA ($r = 0.92$), PFDA ($r = 0.67$), and PFUnDA ($r = 0.70$) but also with PFHxS ($r = 0.29$). There was a significant correlation ($P < 0.001$) between biological age and the sulfonates PFOS ($r = 0.59$) and PFHxS ($r = 0.69$) but also with PFNA ($r = 0.44$). However, the statistical power is low due to the small size of the study group. Age concentrations are in agreement with some studies (*36*, *49*) but not with others (*38*, *41*, *50*). The full information is presented in Table S2 in the Supporting Information.

Correlation coefficients between blood concentrations of different PFCs are given in Table S4 of the Supporting Information. The perfluorinated carboxylic acids PFHpA, PFOA, PFNA, PFDA, and PFUnDA are significantly correlated ($P < 0.001$) to each other and so are PFBA, PFPeA, PFHxA, as well as PFHxS and PFOS. This points to a common exposure, direct or indirect, to the perfluorocarboxylic acids through fluorinated ski wax, since levels are also significantly elevated. Lack of correlation between sulfonates and carboxylates indicates a source of perfluorosulfonates other than ski wax.

An explanation for the wide ranges of elevated carboxylate levels in this study is the number of years in the profession and the human bioaccumulation potential of the compound, which is likely dependent on the chain length.

In conclusion, our study reveals the long half-lives of several carboxylates in humans. We show that PFOA, PFNA, PFDA, PFHpA, and PFUnDA have high bioaccumulation potential and that there is a risk for increasing human body burdens of these perfluorinated carboxylates upon continued lifestyle exposures as well as occupational exposures. Levels may reach far above current background levels for the general population following continued exposure to highly fluorinated consumer products. In future studies, we intend to make qualitative and quantitative assessment of the exposure to airborne fluorinated compounds released during waxing. It also needs to be established if recreational skiers worldwide face increased health risks if exposed to fluorinated ski waxes but also how ski wax degradation products contribute to the environmental pollution.

Acknowledgments

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Supporting Information Available

Tables showing performance of methods and individual blood levels of perfluorinated carboxylates and perfluorinated sulfonates, as well as detailed results of correlation tests. This material is available free of charge via the Internet at http://pubs.acs.org.

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